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Abstract

Melanoma is a cancer of the melanocytes (pigment-forming skin cells) and it takes more lives today than ever before; treatment methods simply cannot keep up with the climbing rate of incidence (1). Although melanoma overall has a 5-year survival rate of 91% and represents only 5% of skin cancers, it is still responsible for 2.7 fold more deaths than all other skin cancers (30). Advancements in the understanding of oncogenic signaling, however, have led to a promising new wave of melanoma treatments (3). These treatments center around BRAF, a gene that when mutated, is believed to be a cause of melanoma. BRAF, like many genes is transcribed into a molecule called mRNA, which is then translated into the functional product, a protein. One of these drugs, Vemurafenib, was associated with a 63% reduction in death when compared to decarbazine (a common chemotherapy drug) in a phase III study (5). Vemurafenib gets its name from its function, V600E mutant BRAF inhibition. Unfortunately, as with most BRAF-V600E inhibitors, Vemurafenib's high initial success rate is followed quickly by resistance to the drug (3). While Vemurafenib acts as a protein kinase inhibitor for the mutated protein product of V600E, the treatment proposed by this paper is to instead target the mRNA of V600E for degradation by siRNA. This treatment will focus on stopping the problem one step earlier in the process than inhibitors such as Vemurafenib. If siRNA is able to knockdown BRAF-V600E, it could be used alongside drugs such as Vemurafenib to improve the current treatment of melanoma.

Honors College
Ball State University
Muncie, IN 47306